ILLUSTRATIONS OF THE ANOMALOUS COURSE OF POSTERIOR SPINAL SCLEROSIS.

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THE typical or normal course of the disease variously known as posterior spinal sclerosis, progressive locomotor ataxia, or more briefly, tabes dorsalis, has become generally known to the profession by the dissemination of recent text-books of diseases of the nervous system and of monographs upon the disease in question. The writings of Charcot and Erb particularly have been of service in thus rendering familiar the numerous symptoms of tabes and their grouping into three stages, viz.: the pre-ataxic or neuralgic stage, second the ataxic stage, and third the pseudo-paralytic or bed-ridden stage.

I prefer grouping the symptoms of typical cases into two stages only, viz.: a neuralgic pre-ataxic stage, and a second or ataxic stage. The so-called third stage is simply an aggravation of the second without any new distinctive symptom.

I do not design to recapitulate the symptoms of posterior spinal sclerosis in this paper, chiefly for the above reason, and also because such a summary is accessible to every reader in Hammond's treatise on diseases of the nervous system, and in a lecture of my own on the "Diagnosis of progressive locomotor ataxia."[1]

Non-typical cases of posterior spinal sclerosis while by no

[1] "Series of American Clinical Lectures," vol. iii., No. 12., N. Y., 1878. Seguin, "Opera Minora," p. 353, N. Y., 1884.

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means rare, are yet often puzzling and misleading. A record of them is desirable, and remarks on their semeiology and probable pathology may not prove wholly devoid of interest.

Numbness, paresis, and ataxia preceding fulgurating pains: antecedent scoliosis and syphilis.

CASE I.—A male æt. 35, a broker by occupation, single, seen in September, 1878. Former health good; from youth has had an extreme right dorsal lateral curvature of the spine caused by persistent over-use of the right arm. Fourteen years ago, when 21 years old, had a chancre followed by some cutaneous eruption, but not by sore throat or osteocopic pains. Remained well until two years ago, when slight numbness appeared in the left foot and leg, and progressively increased. The right foot became involved only in the last few months. During the past summer has noticed progressive weakness of legs, slow micturition, diminution of virile power. Has noticed no abnormal reflexes, spinal pain, cincture feeling, symptoms in hands or head.

Admits no sexual excesses and no injury to spine. The curvature is as it has been for many years.

No neuralgic or fulgurating pains in legs.

The examination showed weak but not ataxic legs; left leg weaker. Marked anæsthesia of soles of feet and toes to æsthesiometer and needle. No muscular atrophy. The patellar tendon reflex and that from the soles of the feet are both lost. No mention of state of pupils.

A thorough anti-syphilitic treatment by means of mercury and iodide of potassium was carried out very carefully by the patient's physician, Dr. T. E. Satterthwaite. This was repeated afterward from time to time.

The consecutive notes of this interesting case are incomplete, but as the patient is still under observation I can give a fairly correct idea of the course of his disease. The paresis passed away in great measure. The numbness and partial anæsthesia remained, extending to the knees. Distinct ataxia, aggravated by closure of eyes, developed in the second year of observation (third of disease), and the first fulgurating pains not till 1880 or 1881, and these were rectal pains, apparently neuralgia of the rectum. Since 1881, occasional pains, sharp, momentary, or burning, in patches, developed coincidently with a low barometer, have occur-

red in the thighs and legs. These have never been severe or long continued; in marked contrast to the usual course of tabes.

The bladder and sexual organs recovered their power and remain normal. This, together with increased firmness in gait, constituting a veritable arrest of the disease, was brought about by nitrate of silver internally, spinal galvanization, and more especially by a course of spinal douches, done under the direction of Prof. Charcot in Paris during 1881.

At no time have the eyes presented symptoms; no diplopia, amblyopia, or Argyll-Robertson pupils. The arms remain normal. During the past winter only a few neuralgic pains have occurred, there was some increase in numbness and staggering, but the ataxia was no greater, and the patient has been able to attend to his business as a broker (office work only) with great regularity.

At the beginning this case presented the clinical picture of a subacute syphilitic myelitis in the lower part of the lumbar enlargement. In its second stage, where it now stands arrested (?) the symptoms consisted in ataxia, partial anæsthesia, absence of patellar reflex, and slight but characteristic fulgurating pains in the rectum and legs. Entirely abnormal to the usual semeiology of tabes are the absence of symptoms about the eyes, and the inverted order of appearance of the pains.

It might be argued that the case was one of central myelitis very low down at first, with secondary changes in the posterior columns, or more properly the columns of Burdach for a certain distance up the lumbo-dorsal cord. Considered in this way, the case (as well as the following) might be taken as favoring the theory that posterior spinal sclerosis is sometimes a secondary and degenerative lesion.

As regards therapy and prognosis the case is interesting. Anti-syphilitic treatment was most clearly called for, and it was carried out very thoroughly, the gums being slightly touched by mercury and iodide of potassium given in quite large doses up to 15. per diem. And the same remedies were

used more than once in smaller doses afterward. The symptoms of myelitis apparently yielded in part to this treatment, but the more strictly tabetic symptoms appeared and persisted. The unmistakable improvement, or check of the disease was obtained later by remedies which are more especially of use in spinal sclerosis, viz.: spinal douches, galvanism and nitrate of silver.

Acute double sciatica, followed by ataxia of the legs.

CASE 2.—A male, aged thirty-two years, married, a merchant by occupation. Seen July 6, 1880.

Was perfectly well until three weeks ago. Had a chancre in 18—. Developed ten days after coition, followed only by "a few pimples."

About June 16th, after a hard day's work in New York calling on many friends, became much heated, and drank freely of cold lemonade. The next day had pain in abdomen and took Congress water without effect. On Tuesday, June 22d, had very severe pain in both sciatic regions, extending to the toes, most acute under the knees ; no numbness. On the 24th came to the city from his country home, with same pain in both sciatic distribution ; a sense of painful pressure and burning in the epigastrium. No loss of power in the legs. Iodide of potassium and salicylic acid were given freely without relieving pains, which were so severe that he had to use laudanum to obtain any rest. The pain was continuous, not stabbing or fulgurating. On the 27th still suffering the same bilateral sciatic pains ; numbness appeared in the feet, rising steadily. On 28th could still walk pretty well, but from the next day (29th) noticed "weakness" of legs ; the bladder acted slowly and imperfectly ; there was pain in the dorsal part of the spine extending into the epigastrium. In the last two or three days the spinal pain has extended to level of the shoulders. The sciatic pain has ceased. The numbness is severe up to knees, and there is slight loss of sensibility of the skin, to level of waist ; a trace of numbness has appeared in the fingers, cannot stand or walk without assistance. Has partial retention of urine and constipation. The muscles of the lower limbs are uniformly though but slightly atrophied. In sleep frequent jerking of legs.

Examination showed some paresis and slight uniform emaciation of the lower extremities. The patient can stand with assistance, but closing his eyes makes this impossible. The bladder is

full of urine, and the catheter draws off three pints. There is neither plantar nor patellar reflex. The chief symptom, however, is typical ataxia of the legs in the attempts to stand and in tests performed while lying on the back.

Without complete anæsthesia, there is marked diminution of sensibility, especially in the right lower extremity. The feet feel numb. No pupillary or other ocular symptoms, and no symptoms in the upper extremities.

The patient had had a chancre some years before, not (?) followed by secondary symptoms; his body is free from cicatrices, and he has two children who are pictures of health. (A third healthy child was presented him by his wife in the year after his attack.)

The patient denied in the most formal manner having had any pains in his legs prior to the attack.

Treatment consisted in the systematic emptying of the bladder twice a day with carbolized catheters; the use of small doses of mercury, and of full doses (up to 5. three times a day) of iodide of potassium, and the application of the galvanic current to the spine and legs. At the end of July was given strychnia.

A peculiar symptom was a severe "griping" pain in the left side of the thorax, from the shoulder forward at the level of the seventh and eighth intercostal spaces. The time of appearance of this pain is not noted, but on July 8th it is "nearly gone," and afterward gave patient very little trouble.

On August 8th the first attack of fulgurating pain is noted, as a stabbing, cutting pain in the internal aspect of the left knee, lasting eight hours.

The case was under observation and treatment two years, during which a certain improvement occurred. The muscles of the legs regained their size and power, the sensibility in a great measure returned, and the bladder (after two attacks of cystitis) recovered its function. But the ataxia remained, though diminished, as the patient was when last seen able to walk with one cane on a sidewalk or piazza several hundred yards, though with characteristic jerk. The fulgurating pains were experienced occasionally during the two years, less frequently, but just as severe and typical. The left side of thorax, though free from pain, was still the seat of a pressure paræsthesia.

This interesting case was quite tabetic in its character after the first eight weeks of its course, the symptoms then being fulgurating pains, ataxia, slight anæsthesia of the legs and

absence of patellar reflex. In its beginning, however, it is quite anomalous, and it is quite probable that the primary lesion was one localized upon the posterior columns of the cord at the end of the seventh or eighth dorsal vertebræ, mostly on the left side of the median line. Such a lesion would account for the severe epigastric and abdominal symptoms experienced at the beginning (dorso-spinal and gastric sympathy is well illustrated in spinal irritation cases), the distinctly localized pain with sense of griping or grasping in the left side of the thorax, in the seventh or eighth intercostal spaces. Such a lesion would also pretty well account for the seeming double sciatica. Many years ago, Cruveilhier showed that *paraplegia dolorosa* was almost pathognomonic of tumor compressing the spinal cord.

This supposed lesion was checked by treatment (?) but not before the conducting power, both for sensibility and for coördination, of the posterior columns had been seriously impaired. In the third week, when first seen, the ataxia of the legs was already extreme. There were also other signs of compression of the dorsal spinal cord, viz.: paresis of the legs and of the bladder (retention). In the seventh or eighth week, and later, the fulgurating pains showed themselves in various parts of the lower limbs, indicating slow sclerotic (or degenerative?) changes in the columns of Burdach below the level of the eighth dorsal nerve.

It is interesting to note in support of the theory of a primary localized lesion that while the thoracic pain was upon the left side, the opposite, right lower extremity was more anæsthetic than the left.

In spite of the absence of secondary symptoms and of transmission of syphilis to offspring and wife, it is possible that the lesion was a specific formation either in the dura mater or more probably in the external layer of neu-

roglia. Its increase was arrested at a certain point, but not before irreparable damage had been done to delicate nerve structures. The question has always been in my mind, would a very active treatment, by means of mercury and large doses of iodide of potassium at the very beginning of the spinal symptoms (fifth day of attack), have saved the patient's legs from permanent disability?

Precocious atrophy of optic nerve; locomotor ataxia.

CASE 3.—Mr. G. C., æt. thirty-six, seen in August, 1878; referred by Dr. Thos. R. Pooley.

About four years ago simultaneous appearance of diplopia, tendency to stagger or walk badly, and specks before eyes. Is positive that at that time and previously he had had no pains in his legs. In the course of a year the diplopia, which was due to paralysis of the left sixth nerve, diminished, but the sight of the left eye failed. From that time vision gradually became worse until lately he has only had perception of light. Dr. Pooley finds extreme atrophy of both optic nerves.

In the last few months patient has had somewhat sharp pains in patches upon the legs and thighs, occurring semi-periodically. The urine has been passed slowly of late. Denies any feeling of numbness in feet, but has had a sensation as of a ball under his feet, and has felt as if treading on rubber. Patient is aware that his staggering is not accounted for by blindness.

Sixteen years ago he had a chancre followed by "warts at the anus," but no other symptoms. Children healthy.

Examination: Optic nerves atrophied, but pupils are "equal and normal" (notes taken at the time). No symptoms in arms. Legs strong but distinctly ataxic; more so when eyes are closed. The soles of the feet are slightly anæsthetic, but pricking is well felt. Absence of patellar reflex. No arthropathies or muscular atrophy.

This case belongs to a category which I suspect is not small. At least I have met with several such in which the amaurosis was coincident with or antecedent to the ataxic stage. At the present time there are two cases under my care in which, with very little ataxia, there exists complete loss of vision, in one case with atrophy of the optic nerves, in the other with slight ophthalmoscopic signs.

What is interesting to note is that in such cases we have not to do with an extended longitudinal sclerosis (at least judging by clinical signs), for in these three cases the arms remained normal at the time of last examination. The lesions in the optic apparatus and those in the dorso-lumbar cord doubtless arise under the pathogenetic law of sclerosis, which at present is wholly unknown to us. Cases of this variety, in which the optic apparatus suffers only, and the more numerous cases in which paralysis of one or more ocular muscles precedes (sometimes by years) the first or neuralgic stage of tabes, constitute an almost insuperable objection to the theory that posterior sclerosis is a degenerative lesion secondary to disease of peripheral nerves—an ascending degeneration.

Extraordinary prolongation of the pre-ataxic or neuralgic stage (29 *years*).

Case 4.—In 1878 I was consulted by an artist, 57 years of age, for a "neuralgia," which had tormented him from his thirtieth year. The pains affected the lower extremities only until two or three years ago, when they showed themselves in the arms as well. Patient describes these pains as sudden, sharp, tearing, sometimes of atrocious severity, occurring in spots or patches of round or oblong outline. These pains recur in one spot for some time, varying from a few minutes to hours and days. In the course of these many years he has had foci of pain in nearly every part of the lower extremities, more especially near the knees and ankles. In the last few years the intervals between paroxysms have become shorter, and the pains have grown more severe. There is now mydriasis of the right eye, a condition which has existed thirty years without diplopia. A mere trace of numbness has made its appearance in the legs, detected only at times by rubbing the skin. The painful spots are hyperæsthetic during the paroxysms. In the last few years the urine has passed slowly. The floor or ground feels normal under foot; no difficulty in walking. Examination shows dilatation of the right pupil, without diplopia; no changes in the optic nerve (patient worked at his art to the last). No ataxia of the upper and lower extremities. There is very slight staggering when patient attempts to stand with eyes

closed. The soles of the feet show slight anæsthesia to æsthesiometer test. Reflex from ligamentum patellæ lost. No paresis.

About two years after this first examination, Mr. A. died of a combination of renal and cardiac diseases. His spinal symptons had remained unchanged ; no ataxia at any time.

The diagnosis of posterior spinal sclerosis was verified in the post mortem examination of the spinal cord ; a distinct sclerosis of the lateral parts of the posterior columns was found.

Prolonged neuralgic stage (12 *years*)*; early arthropathies.*

CASE—5. A male patient sent to my clinic in 1877(?) by Dr. C. Williams. Has had characteristic pains in the legs for 12 years ; spots of pain hyperæsthetic at time of attack. Slight numbness of feet ; swelling in both knees in last two years ; no difficulty in locomotion.

Examination shows moderate anæsthesia and analgesia in feet and legs to knees ; absence of patellar tendon reflex. Chronic arthritis of both knee-joints with crepitations. Careful tests with eyes open and closed revealed no staggering or ataxia.

Prolonged first stage; development of general paralysis of the insane before ataxia.

CASE 6. An ex-army officer seen in July 1878. He was then 35 years of age and apparently in good health. Was much exposed during the war in 1864–5, and in the latter year had typho-malarial fever. Then enjoyed better health for several years. In 1876, while on duty in the far West, had two attacks of "cerebral congestion," in one of which he fell unconscious. This was followed by poor health, depression, and hypochondriasis ; was almost insane on the subject of abuse by brother officers, official neglect, etc. Left the army and seemed well again, though a strong feeling of professional disappointment was prominent in his mental state.

Mr. B. consulted me for a peculiar "neuralgia." As far back as 1872 he had had attacks of cutaneous pain, becoming more and more frequent and severe. Attacks irregular as to time and location, mostly below the waist ; a few lately in the arms. Pain is sharp, cutting, and tearing, affecting round or oval areas in the skin and subjacent muscles ; not at all in the course of nerve-trunks. The pains were at times extremely severe, and their seat was nearly always hyperæsthetic.

Has had no numbness, vesical or optic symptoms. No sexual excitement.

Examination showed absence of patellar tendon reflex, and a little staggering when standing with eyes closed.

In 1881 I met Mr. B., but under such circumstances that I could not examine him. I was able to observe, however, that his pupils were extremely small. He seemed to stand well during the conversation.

During 1883–4 the case rapidly developed into a very well-marked one of general paralysis, with exaltation. The following details were obtained from Mrs. B.:

Excepting for attacks of fulgurating pains Mr. B—— seemed fairly well until 1882. During the winter of 1882–3 he was often depressed, and was oftentimes found weeping and sobbing in his library. At other times was bright, hopeful, and even "high" without actual delusions. Of these abnormal psychic states the depressed periods were longer and more marked. During the summer of 1883 delusions and exaltation appeared. At times considered his wife insane; at others blamed her for his illness, because of refusal to have complete sexual intercourse. After he was placed (July, 1883) in the private institution where he now lives, he declared that his wife had feigned his insanity to get him incarcerated! Letters written in November, December, 1883, and January, 1884, are typical of general paresis in composition and mechanical execution. I saw him in February and he remembered me perfectly, as also my diagnosis of posterior sclerosis. He laughed at this, and stiffened out his leg and arm, and strutted about to show me how absolutely free from ataxia he was. He appeared prematurely aged; his speech was quick and quivering; his facial and lingual muscles showed fibrillary tremors. His walk was slightly ataxic, and there was no patellar reflex. The pupils were unequal in size and did not respond to light and shadow. During the past spring the patient had two epileptoid attacks, with semi-coma. This was followed by temporary right hemiplegia and aphasia. Later he was reported as quiet, childish, and full of exalted notions.

From 1872 to 1882, a period of eleven years, the chief symptoms in this interesting case were the fulgurating pains, absence of tendon reflex, and fixed pupils (?). The ataxia observed during the present year is hardly as distinct as we see it in ordinary tabes, and the walk resembles that of general paretics.

Cases of tabes terminating with symptoms of general paralysis are not very rare, but in all I have seen, the spinal symptoms were complete and advanced before signs of cerebral degeneration showed themselves. In the case related it seemed as if the dependence between the cerebral and the spinal lesion was doubtful. From what we know of the pathological anatomy of the two affections, it would seem that Mr. B—— had only a slight sclerosis of the columns of Burdach (the posterior radicular zones) at the same time that his cortex cerebri was the seat of advanced and rapidly progressing inflammatory action.